Information

CLINICAL CARDIOLOGY SERIES

Cardiac Tamponade and Constrictive Pericarditis

Part II—Treatment

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In the preceding article in this series, pathophysiology, clinical signs and symptoms, and etiologic feature of cardiac tamponade due to pericardial effusion and constrictive pericarditis were discussed. We shall now consider treatment of these conditions.

Tamponade

The only treatment for tamponade is removal of the fluid. If the patient is moribund, pericardiocentesis is carried out as an emergency measure. More often, the patient should be moved to the operating suite, intensive care area or cardiac catheterization laboratory. Of the several approaches to the pericardium, the xiphisternal is the safest. The patient rests supine with the thorax elevated 45 degrees. The skin and subcutaneous tissues a few mm from the xiphisternum are cleansed and infiltrated, and then pierced with the tip of a No. 11 blade. An 18T needle is advanced posteriorly until its tip has negotiated the bony margin of the thorax. The needle is then advanced close to the midline with a slight posterior tilt, until the pericardium is reached. If there is much effusion, little or no cardiac pulsation will be felt. The needle is advanced through the pericardium, usually with a palpable sensation.

A conventional electrocardiogram lead should be monitored throughout the procedure. If it is absolutely certain that all equipment is properly grounded, the position of the needle tip can be monitored by a direct ECG lead. A cable connects the needle hub and an indifferent electrode (for example, a limb lead) to the electrocardiograph which is set in position "V." If the needle tip touches the epicardium a large ST segment elevation occurs in the needle lead, and a smaller one in the conventional lead. Also ventricular extrasystoles may be provoked. When these phenomena occur, the needle should not be advanced further, but should be withdrawn slowly and carefully until the ST segments are again isoelectric.

As soon as the pericardium is entered, a specimen is aspirated; and if it is bloody, its hematocrit and clotting time are compared with these features of the patient's blood. Hemorrhagic fluid rarely clots and almost always has a lower hematocrit than venous blood. Pericardial pressure should then be measured with a manometer, after which specimens should be obtained for bacteriologic, chemical and cytologic studies. Removal of the first 50 to 200 ml of fluid usually produces prompt relief because of the steep pericardial pressure volume curve. Venous pressure and pulsus paradoxus decrease and the arterial pulses become full. Pericardial fluid is aspirated as long as it can be withdrawn easily, and preferably until the pericardial pressure is zero or less. No elaborate precautions to prevent air aspiration are needed; indeed, introduction of a volume of air or CO_2 equal to less than half the aspirated fluid volume is useful to delineate the pericardium in subsequent chest roentgenographs.

Sometimes it is desirable to leave a small tube in the pericardium. Chemotherapeutic agents may be instilled into the pericardial sac. A chest roentgenograph is obtained at the end of the procedure. Appropriate systemic antibiotic therapy is commenced, and if tuberculosis is a reasonable possibility, steroids may be administered as well.

If a delay in initiating pericardiocentesis is inevitable, the circulation can be temporarily sustained by infusion of isoproterenol together with volume expansion by means of dextran blood or saline solution. Isoproterenol is started at 2 to 4 micrograms per minute and increased until perceptible effects on the pulse and pressure are

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evident. Recurrence of tamponade may require repetition of pericardiocentesis, but surgical drainage may then be preferable. In traumatic cases, recurrence after one or two successful aspirations is an indication for surgical drainage. When tissue diagnosis is important and a physician experienced in the technique of pericardiocentesis is not available, the pericardium can simply be drained and a biopsy specimen taken under local anesthesia after excision of the xiphoid process.

Constrictive Pericarditis

Once a diagnosis of constrictive pericarditis is established, the patient should be referred to a thoracic surgeon for pericardiectomy. Patients improve following digitalization because of the frequent myocardial involvement associated with the constriction. Tachycardia secondary to atrial fibrillation responds well to digitalis, as in heart failure. These measures alone produce diuresis, but it is usually advisable to administer diuretic agents as well. These are given in high dosage for as long as they are effective and serum sodium and chloride levels are not excessively depressed. Serum potassium must be maintained at normal levels by means of oral potassium chloride administration, and salt intake should be moderately reduced. Because of liver congestion, alcohol and other hepatic toxins must be forbidden. These measures may be employed to effect an immediate improvement and to prepare the patient for pericardiectomy. They are also remarkably successful for variable periods in patients who are unwilling to undergo operation, but in these circumstances slow progression of physiologic decompensation invariably occurs.

Pericardiectomy is now a safe operation. In a young person without pericardial calcification, cardiac enlargement, or atrial fibrillation the mortality is virtually zero. Thus the operation is urgently indicated in subacute constrictive pericarditis. In chronic calcific constrictive pericarditis, the operative risk with experienced cardiac surgeons is 5 to 10 percent. Pericardiectomy is not an open heart procedure, but full cardiopulmonary bypass facilities should be immediately available to deal with cardiac trauma, evidence of which may appear suddenly at any time during the operation.

Several months elapse after operation before recovery is complete, especially in the more chronic cases. Venous pressure is reduced somewhat immediately following operation but only slowly returns to normal. Digitalis and diuretic agents may be required for several months to control edema and effusions. In the most chronic cases with heavy calcification and long standing congestion, the aim is improvement rather than cure. The results in such cases are limited by myocardial involvement, hepatic insufficiency, chronic pulmonary congestion and pleural disease.

Cardiac compression secondary to malignant disease should be managed as conservatively as possible. Pericardiocentesis and the instillation of cancer chemotherapeutic agents may control the process in some patients. In others it may be necessary to construct a pericardiopleural window to drain the pericardium via the pleural space. There is little to be done for patients with constrictive pericarditis secondary to malignant disease without an element of pericardial effusion. Surgeons are generally appropriately reluctant to perform pericardiectomy in such circumstances.